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Increased muscle responses to balance perturbations in children with cerebral palsy can be explained by increased sensitivity to center of mass movement

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ABSTRACT

Background: Balance impairments are common in children with cerebral palsy (CP). Muscle activity during perturbed standing is higher in children with CP than in typically developing (TD) children, but we know surprisingly little about how sensorimotor processes for balance control are altered in CP. Sensorimotor processing refers to how the nervous system translates incoming sensory information about body motion into motor commands to activate muscles. In healthy adults, muscle activity in response to backward support-surface translations during standing can be reconstructed by center of mass (CoM) feedback, i.e., by a linear combination of delayed (due to neural transmission times) CoM displacement, velocity, and acceleration. The level of muscle activity in relation to changes in CoM kinematics, i.e., the feedback gains, provides a metric of the sensitivity of the muscle response to CoM perturbations.
Research question: Can CoM feedback explain reactive muscle activity in children with CP, yet with higher feedback gains than in TD children?
Methods: We perturbed standing balance by backward support-surface translations of different magnitudes in 20 children with CP and 20 age-matched TD children and investigated CoM feedback pathways underlying reactive muscle activity in the triceps surae and tibialis anterior.
Results: Reactive muscle activity could be reconstructed by delayed feedback of CoM kinematics and hence similar sensorimotor pathways might underlie balance control in children with CP and TD children. However, sensitivities of both agonistic and antagonistic muscle activity to CoM displacement and velocity were higher in children with CP than in TD children. The increased sensitivity of balance correcting responses to CoM movement might explain the stiffer kinematic response, i.e., smaller CoM movement, observed in children with CP.
Significance: The sensorimotor model used here provided unique insights into how CP affects neural processing underlying balance control. Sensorimotor sensitivities might be a useful metric to diagnose balance impairments.

1. Introduction

Balance impairments are common in children with cerebral palsy (CP) [1–4], but we know surprisingly little about how sensorimotor processes for balance control are altered in CP. Sensorimotor processing refers to how the nervous system translates incoming sensory information about body motion into motor commands to activate muscles. We know that both kinematic and muscle responses to perturbations of standing balance differ between children with CP and typically developing (TD) children [1,3,5–12]. When standing is perturbed by backward support-surface translations, children with CP step forward at lower perturbation velocity [10,12,13]. Antagonistic muscle activity (i.e., tibialis anterior) – and thus muscle co-activation - is higher in children with CP than in TD children [5,8]. In addition, children with CP do not modulate reactive muscle activity amplitude with perturbation amplitude, as seen in TD children and healthy adults [6,14]. However,

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we still do not understand the sensorimotor processing deficiencies leading to changes in balance control in children with CP.

In both healthy and pathological animals and humans, sensorimotor processes underlying reactive standing balance can be explained by delayed (due to neural transmission times) feedback of center of mass (CoM) kinematics [15–18]. In other words, muscle activity in response to a perturbation of standing balance can be described by a linear combination of delayed CoM displacement, velocity, and acceleration. This suggests that the central nervous system combines information from different sensors (including visual, vestibular, proprioceptive, and cutaneous) to estimate CoM kinematics and translates this information into motor commands, a process called sensorimotor transformation. The weights or gains of the CoM displacement, velocity, and acceleration in the linear combination indicate the sensitivity of the muscle response to the CoM disturbance. These sensitivities have been shown to be relatively constant within one subject [16] but change with age [17, 18], age-related cognitive decline [17], sensory deficits [15] and Parkinson's disease [18]. Hence, CoM feedback pathways were preserved across a range of neurological conditions while CoM feedback gains might reflect the underlying condition.

Given the pathophysiology, the increased muscle co-activation and lack of modulation of muscle responses during perturbed standing in children with CP, we predicted increased CoM feedback gains for both the agonistic and antagonistic muscles. Spasticity is the most common symptom in CP. Decreased inhibitory supraspinal control in both passive and active muscles [19,20] leads to stretch hyper-reflexia [21]. Children with CP are thus less able to adequately modulate stretch-induced muscle responses [20]. Clinically, spasticity presents as increased resistance to passive muscle stretch. When standing balance is perturbed, muscles are stretched as well and therefore a similar lack of inhibition might lead to an increased muscle response. Reduced selective motor control is another common impairment in CP and refers to a reduced ability to activate muscles or muscle groups in isolation [22]. Reduced selective motor control might thus lead to co-activation between agonistic and antagonistic muscles.

Here, we investigated sensorimotor transformations underlying the response to backward support-surface translations during standing in children with CP. We hypothesized that reactive muscle activity in both children with CP and TD children could be explained by delayed feedback of CoM kinematics but that feedback gains for both agonistic and antagonistic muscles would be higher in CP. We applied backward support-surface translations of six different levels (increasing platform displacement, velocity, and/or acceleration) to 20 children with CP and 20 age-matched TD children. We evaluated the fit between measured reactive activity of the gastrocnemii, soleus, and tibialis anterior and reconstructions based on CoM feedback as well as differences in feedback gains between groups.

2. Methods

2.1. Subjects

The Ethical Committee of UZ/KU Leuven (S63321) approved the study. Forty-six children participated and signed an informed assent form whereas their legal representative signed an informed consent form following the Declaration of Helsinki. Children with spastic CP were diagnosed by a neuro-pediatrician and met the following inclusion criteria: (1) 5–17 years; (2) Gross Motor Function Classification Scale (GMFCS) I-III; (3) able to stand independently for at least 10 min; (4) no orthopedic/neurological surgery in the previous year; and (5) no Botulinum Toxin injections in the previous 6 months. TD children were age-matched with children with CP. Data from six children were excluded due to (1) incomplete protocol (N = 1), (2) problems with following instructions (N = 3) or (3) technical problems (N = 2).

Data from 20 TD children (8 girls) and 20 children with CP (9 girls) were analyzed (Table 1). Fourteen and six children with CP were respectively unilaterally and bilaterally involved. Fifteen and five children had respectively GMFCS level I and II. Sixteen children had gastrocnemius spasticity, as indicated by a Modified Ashworth Scale (MAS) between 1 and 3 (supplementary table S1).

Table 1
Demographic data of participants.

		CP			TD		
		Mean	*SD*	*Range*	*Mean*	*SD*	*Range*
Girls/ boys		9/11			8/16		
Age	**(years)**	12.3	3.1	7–17	11.5	3.4	7–17
Length	**(cm)**	153	16	130 – 185	154	18	126–184
Weight	**(kg)**	47	17	27–88	42	14	25–67
Hemi- / di-plegic		14/6					
GMFCS I / II		15/5					
MAS 0/1/1 + /3		4/10/4/ 2					

CP = cerebral palsy; TD = typically developing; GMFCS = Gross Motor Function Classification Scale (range 1-5); MAS = Modified Ashworth Scale (range 0-4). No significant differences in age, length, or weight between children with CP and TD children.

For children with CP, the most affected leg (based on MAS) was analyzed, while for TD children one leg was randomly selected.

2.2. Protocol

Reactive balance was tested on a Caren platform (Motek, The Netherlands) (Fig. 1a). Trajectories of reflective skin markers (whole body marker set, Fig. S1) were captured by 7 infrared Vicon cameras at 100 Hz (Oxford Metrics, United Kingdom). Activity of lateral gastrocnemius (LG), medial gastrocnemius (MG), soleus (SOL), and tibialis anterior (TA) was measured simultaneously through surface electromyography (EMG) at 1000 Hz (ZeroWire EMG Aurion, Cometa, Italy). Electrodes (Ambu Blue Sensor, Ballerup, Denmark) were placed according to SENIAM guidelines [23]. Participants stood barefoot on the platform (starting position was marked and consistent across trials) and were secured with a safety harness. Instructions were to stand upright and to maintain balance without stepping unless stepping was necessary to avoid falling. Arm movement was unconstrained. The protocol consisted of six increasingly difficult perturbation levels (increased platform displacement, velocity and/or acceleration, Fig. 1b) [24]. Within each level, eight perturbations were administered (12 s between perturbations, about 2 min per level). When participants stepped in more than 3 trials within a level, we did not continue to the next level. If needed, rest was given between levels.

2.3. Data processing & analysis

Marker trajectories were processed using OpenSim 3.3 [25,26]. A generic musculoskeletal model (full body - Hamner 2010) was scaled based on anatomic marker positions [27,28]. CoM position was computed by consequently applying OpenSim's Inverse Kinematics and Body Kinematics Tools. CoM displacement relative to the ankle was numerically differentiated to compute CoM velocity. CoM acceleration was computed using a Savitzky-Golay filter [29]. For each participant and level, average CoM kinematics were calculated across all non-stepping trials.

EMG data was filtered with a fourth order Butterworth band-pass filter with 10 and 450 Hz cut-offs, rectified, and filtered with a fourth order Butterworth low-pass filter with 40 Hz cut-off [30]. The filtered EMG signal was scaled by the maximum value across all perturbations, including toe-up rotational perturbations performed as part of a larger protocol. The average EMG signal was calculated across all non-stepping trials for each participant and level.

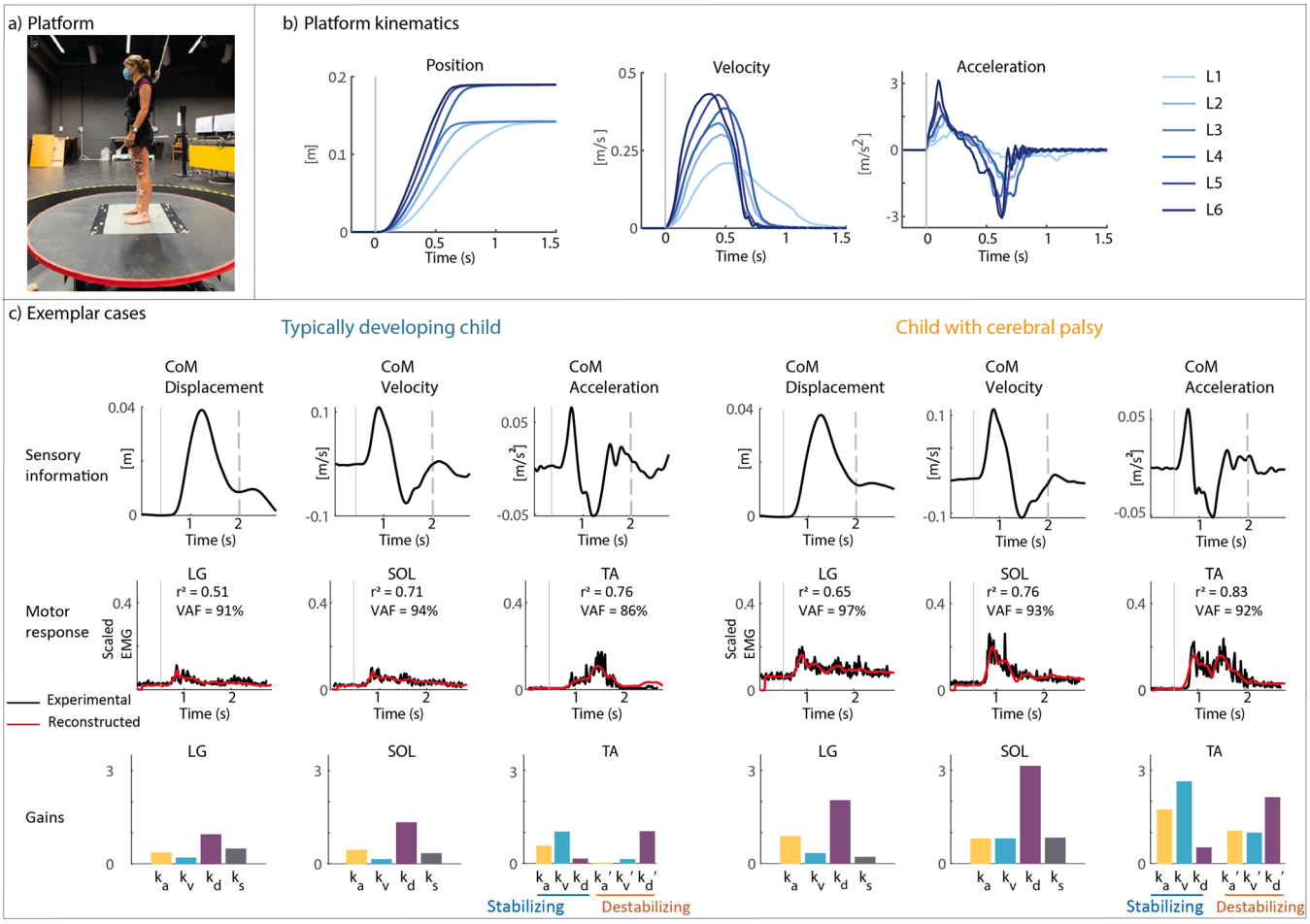

Fig. 1. Reactive balance test set-up and exemplar responses. a) Platform for backward translations. b) Platform position, velocity, and acceleration profiles for the different perturbation levels (L1-L6). c) Exemplar cases for one typically developing child (left) and one child with cerebral palsy (right). Center of mass kinematics (top row), experimental (black) and fitted (red) muscle responses of lateral gastrocnemius (LG), soleus (SOL) and tibialis anterior (TA) (middle row), subject specific estimated feedback gains (k_a, k_v, k_d, k_s) and prime gains (k_a', k_v', k_d') (bottom row). Maximum CoM displacement was calculated over the time interval indicated by the full and dotted grey line.

2.4. Outcome parameters

We assessed the *kinematic strategy* by evaluating the **number of completed levels**, representing the use of a non-stepping strategy, as well as CoM movement during non-stepping responses. We computed the **maximal horizontal CoM displacement** in a period of 1.5 s following perturbation onset as the maximum CoM displacement was reached during this period (dotted line, Fig. 1c).

To evaluate *sensorimotor transformations*, we reconstructed measured EMG trajectories by delayed feedback from CoM kinematics. At perturbation onset, the platform accelerated backward and the body swayed forward, eliciting a balance-correcting response in the triceps surae. We used the following sensorimotor response model to reconstruct triceps surae activity (Figs. 1c-2) [15,16]:

$$EMGrecon = e_0 + \lfloor k_d * d_{CoM}(t-\tau) + k_v * v_{CoM}(t-\tau) + k_a * a_{CoM}(t-\tau) + k_s * a_{CoM\_Init}(t-\tau) \rfloor \quad (1)$$

with EMG_{recon} reconstructed muscle activity; e_0 baseline muscle activity (i.e., activity during quiet standing); d_{CoM}, v_{CoM}, and a_{CoM} respectively CoM displacement, velocity, and acceleration; k_d, k_v, k_a feedback gains or weights, and τ a common time delay of 100 ms to account for processing and neural transmission time. We added a separate feedback term for the initial CoM acceleration, a_{CoM_Init}, with a corresponding stiction gain k_s inspired by Welch and Ting [16]. The initial burst in EMG is proportional to the initial CoM acceleration [16] and might be driven by the initial strong increase in spindle firing coinciding with short-range stiffness in the muscle [31]. We therefore included this term until the change in ankle angle was 0.5°, corresponding to the plantarflexors' estimated short-range stiffness range [32,33]. In contrast to Welch and Ting, we included both an acceleration and stiction term based on preliminary analyses. Only the positive part of the signal (indicated by $\lfloor\rfloor$) was used to represent excitatory drive to motor pools.

Especially in CP, we often observed a large burst of TA activity upon acceleration of the platform, opposing the balance correcting activity of the plantarflexors. We therefore used a more complex sensorimotor response model for TA that could capture both its role as an antagonist when the platform accelerated (destabilizing pathway, Fig. 2) and as an agonist when the platform decelerated at the end of the perturbation (stabilizing pathway, Figs. 1c-2)[18]:

$$EMGrecon = e_0 + \lfloor k_d * -d_{CoM}(t-\tau) + k_v * -v_{CoM}(t-\tau) + k_a * -a_{CoM}(t-\tau) \rfloor (Stabilizing\ pathway) + \lfloor k_d' * d_{CoM}(t-\tau) + k_v' * v_{CoM}(t-\tau) + k_a' * a_{CoM}(t-\tau) \rfloor (Destabilizing\ pathway) \quad (2)$$

with k_d', k_v', k_a' prime gains of the destabilizing pathway.

Baseline activity was set to the mean of the experimental muscle activity 0.5 s before perturbation onset. Gains were estimated by

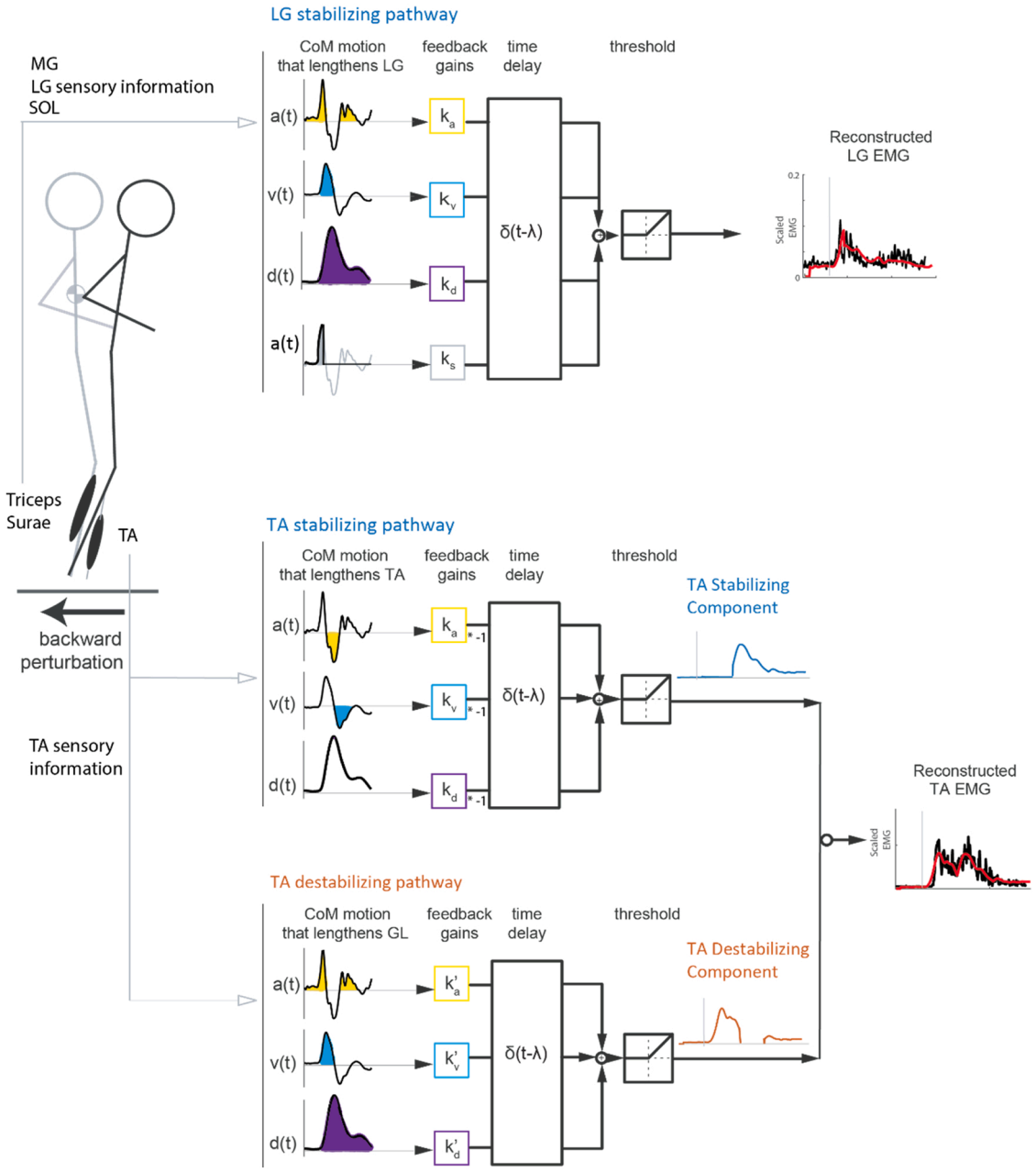

Fig. 2. Reconstruction of experimental muscle activity using delayed feedback of CoM kinematics (example for one triceps surae muscle, i.e., lateral gastrocnemius (LG), and tibialis anterior (TA)). Sensory information signals, CoM acceleration (yellow), velocity (blue), and displacement (purple), related to backward support-surface translations are multiplied by subject specific feedback gains (k_a, k_v, k_d,). The initial CoM acceleration trajectory (grey) multiplied with a stiction gain (k_s) was used to account for the short-range stiffness response in the plantarflexors (MG = medial gastrocnemius; SOL = soleus). Scaled kinematic signals are then delayed and summed to reconstruct experimental muscle activity (black). Optimized feedback gains are found by minimizing the difference between experimental and reconstructed (red) muscle activity signals. For the TA, muscle activity was reconstructed using a stabilizing pathway with gains (k_a, k_v, k_d,) (when the platform decelerates) and a destabilizing pathway with prime gains (k_a', k_v', k_d') (when the platform accelerates).

minimizing (fmincon, Matlab R2018b, Mathworks, United States) the squared difference between reconstructed and measured activations over a time interval from 0.5 s before until 1.5 s after perturbation onset. Displacement, velocity, and acceleration gains were constraint between 0 and respectively 10/m, 10 s/m, 10 s^2/m [14].

To test whether CoM feedback can explain reactive muscle activity, we assessed the goodness of fit between predicted and reconstructed muscle signals using both the **coefficient of determination (r^2)** and the **variability accounted for (VAF)** [14]. To test how sensitive reactive muscle activity was to CoM perturbations, we assessed the **gains**. Gains indicate the sensitivity of the muscle response to CoM perturbations. For example, a higher displacement gain, and prime displacement gain for TA indicates higher muscle activity for respectively the same backward and forward CoM displacements. In addition, we compared mean **reactive muscle activity** between groups (Supplement S6).

2.5. Statistical analysis

All statistical analyses were performed using Matlab with differences considered significant at $p < 0.05$.

A Kolmogorov-Smirnov test indicated that data was non-normally distributed.

Differences in maximal CoM displacement between groups were tested using a linear mixed model with two fixed effects: (1) group (CP vs. TD) and (2) perturbation level (1–6, ordinal). A participant factor was included as random factor nested within group. Observation entered in models were the average per participant across non-stepping trials within one perturbation level. We expected that more impaired children with CP would only be able to perform lower levels of perturbations resulting in larger differences in CoM displacement between groups in the lower than in the higher levels. Therefore, we tested the difference between groups (1) across all perturbation levels and (2) across perturbation levels 1–3.

Differences in r^2 and VAF between groups were tested (across all perturbation levels for each muscle) using a Mann-Whitney U test.

Differences in gains between groups were tested using a linear mixed model as described above. The mixed model was applied four times (k_a, k_v, k_d, k_s) for LG, MG, and SOL and six times (k_a, k_v, k_d, $k_a^{'}$ $k_v^{'}$, $k_d^{'}$) for TA. All tests for model parameters (gains) for each muscle were performed without adjustment of simultaneous interference as they were assumed to evaluate independent hypothesis.

3. Results

Due to technical errors in EMG recordings, we had to exclude data for LG in 1 child with CP, MG in 1 child with CP and 1 TD child, SOL in 1 child with CP, and TA in 2 children with CP.

3.1. Kinematic strategy

Children with CP performed fewer levels without stepping than TD children (Fig. 3a). Only one TD child did not perform levels 3–6. Two children with CP did not perform levels 2–6, one child did not perform levels 3–6, two children did not perform levels 4–6, and five children did not perform levels 5–6.

Children with CP had a smaller maximal CoM displacement compared to TD children in level 1 ($p = 0.007$, median CP: 0.032 m; TD: 0.039 m), level 2 ($p = 0.016$, median CP: 0.055 m; TD: 0.062 m) and level 3 ($p = 0.009$, median CP: 0.063 m; TD: 0.074 m) for non-stepping trials when only considering perturbation levels 1–3 in the statistical analysis. No differences were found between the two groups when including all perturbation levels (Fig. 3b, table S2).

Children with CP had higher reactive muscle activity than TD children (S6, table S5, Fig. S4).

3.2. Sensorimotor processing

CoM feedback could explain muscle activity in both groups as reflected in the high goodness of fit values (r^2 – CP: 0.71 ± 0.12, TD: 0.67 ± 0.15, Fig. 4a; VAF – CP: 90.3% ± 4.8, TD: 89.14% ± 6.0, Fig. 4b). Goodness of fit values across all levels did not differ between groups for MG and SOL ($p > 0.05$). Goodness of fit values were higher in LG (r^2: $p = 0.008$) and TA (r^2: $p = 0.001$; VAF: $p < 0.001$) for children with CP than for TD children (Fig. 4, table S3).

Children with CP had higher displacement and velocity feedback gains than TD children (Fig. 5, Figs. S2-S3, table S4).

For LG (Fig. 5a), velocity ($p < 0.001$) and displacement ($p < 0.001$) gains were respectively 108% and 104% higher in children with CP compared to TD children across all levels. Furthermore, there was an interaction effect between level and group for the displacement gain

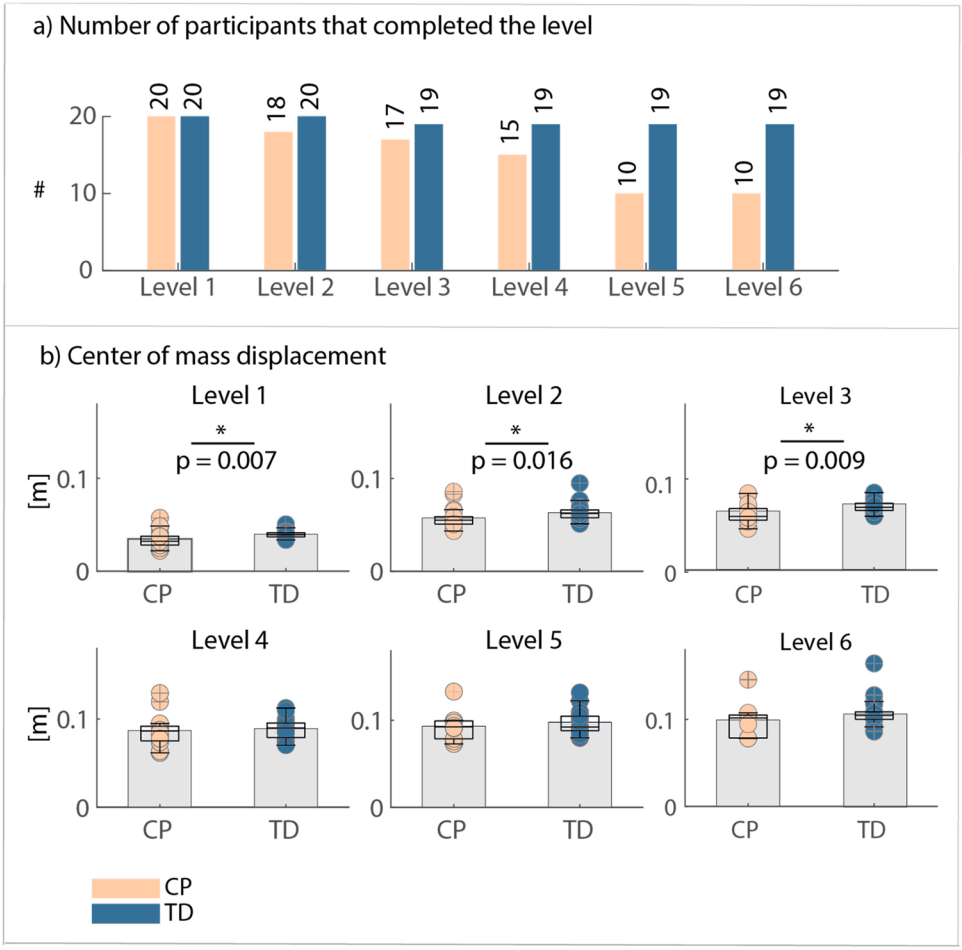

Fig. 3. Kinematic strategy. **a)** Number of participants that completed the level. **b)** CoM displacement in the horizontal plane with respect to the ankles for every level. Grey bars indicate group averages, boxplots in black indicate median and interquartile range, dots are individual scores. Children with cerebral palsy in orange (CP), typically developing children in blue (TD). Significant differences (based on the mixed model that only considered perturbation levels 1–3, $p < 0.05$) are indicated with a star and p-values.

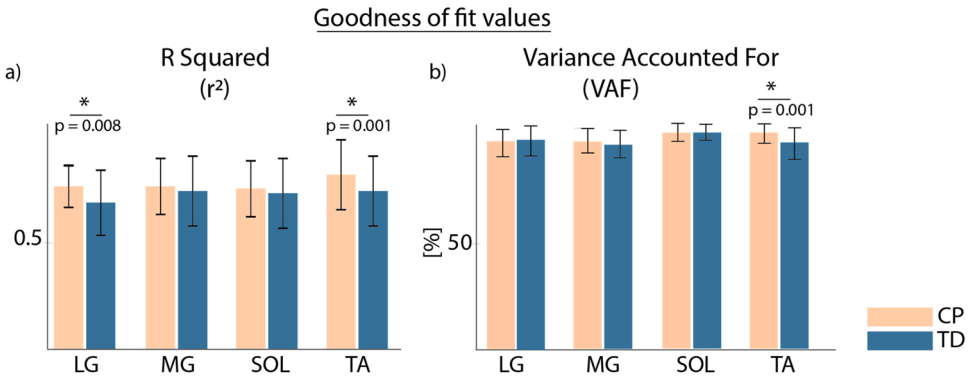

Fig. 4. Goodness of fit values averaged across all levels. **a)** r squared, **b)** variance accounted for values. Children with cerebral palsy (CP) in orange, typically developing (TD) children in blue. Significant differences ($p < 0.05$) are indicated with a star and p-values. LG = lateral gastrocnemius; MG = medial gastrocnemius; SOL = soleus; TA = tibialis anterior.

($p = 0.046$), suggesting larger differences between both groups for the lower levels (visual exploration).

For MG (fig. S2), the velocity gain ($p = 0.034$) was 41% higher in children with CP compared to TD children across all levels.

For SOL (fig. S3), velocity ($p = 0.001$) and displacement ($p < 0.001$) gains were respectively 42% and 28% higher in children with CP compared to TD children across all levels. Furthermore, there was an interaction effect between level and group for the displacement gain ($p = 0.01$).

For TA (Fig. 5b), velocity ($p = 0.02$), prime velocity ($p = 0.05$) and prime displacement ($p = 0.03$) gains were respectively 54%, 71%, and 103% higher in children with CP compared to TD children across all levels. Furthermore, there was an interaction effect between level and group for prime velocity ($p = 0.009$).

Gains decreased with increasing perturbation level for all muscles ($p < 0.05$, table S4), except for the stiction gain in the gastrocnemii and the acceleration, displacement, and prime acceleration gains in TA.

4. Discussion

Similarly as in healthy and pathological animals and human adults [15–18], delayed CoM feedback can explain motor responses to backward support-surface translations during standing in TD children and children with spastic CP. Children with CP might thus use similar sensorimotor pathways as TD children for balance control. Yet, displacement and velocity feedback gains in both agonistic and antagonistic muscles were higher in children with CP than in TD children. The feedback gains provide a metric for the sensitivity of the muscle response to CoM perturbations [15,16], whereas it is hard to distinguish differences in kinematic perturbations and alterations in the response when evaluating muscle activity alone. Increased sensitivity to CoM movement across agonists and antagonists might explain the stiffer response (smaller CoM displacement) to balance perturbations observed in children with CP and possibly the need to step at much lower perturbations, that has also been reported previously [10,12,13].

The increased sensitivities for CoM displacement and velocity in both the stabilizing and destabilizing pathways offer an explanation for the increased reactive muscle activity and muscle co-activation that has been reported previously [1,5,8,10,34]. The magnitude of the sensitivities depends on EMG scaling. Higher activations across perturbation conditions in children with CP than in TD children might therefore have led to an underestimation of the difference between both groups. The lack of differences in acceleration gains between groups might be due to lower platform accelerations as compared to other studies [14,18]. In Parkinson's disease, increased TA sensitivity to CoM acceleration in the destabilizing pathway is correlated with falling [18]. Future studies should collect information on fall history to investigate whether sensitivities in children with CP also correlate with falling. Although antagonistic muscle activity is increased in both CP and Parkinson's disease, increased agonistic muscle activity was not observed in Parkinson's disease suggesting disease-specific alterations in neural processing.

Our observations do not confirm the previously reported lack of modulation of the muscle response amplitude to perturbation level in CP [5,6]. There was no interaction effect for mean plantarflexor activity between perturbation level and group (table S5), indicating that both groups increased their muscle activity similarly with perturbation level. TA activity increased even more between levels in children with CP than in TD children (fig. S5). The interaction effect for MG/SOL displacement gains and TA prime velocity gain suggesting larger differences in gains between groups for the lower levels might be a result of the participants with poorer balance control not performing the higher levels.

Our results suggest that reduced CoM displacements in response to a perturbation should not be interpreted as a sign of better balance control. Children with CP had stiffer responses, i.e., reduced CoM displacement, than TD children for the milder (perturbation levels 1–3) but not for the stronger perturbations. Children who had poorer balance control and were not able to perform the lower perturbation levels without stepping did not proceed to the higher levels. The differences in CoM displacements for the milder but not for the stronger perturbations thus indirectly suggest that children with stiffer balance responses had poorer balance control. The stiffer response might have been caused by higher muscle co-activation increasing joint impedance. We indeed found that higher TA feedback gains for the destabilizing pathways explained lower CoM displacements (supplement S3). It is unlikely that differences in anticipatory movements (constant perturbation direction and timing) contributed to differences in maximal CoM displacements between both groups as CoM position and baseline activity at perturbation onset was not different between groups, and subjects did not anticipate perturbation onset (Supplement S8, table S6-7). Yet, we found differences in baseline TA activity between groups (Supplement S9, table S8).

The highly variable gains in children with CP (visual inspection) reflect the heterogeneity in how the disease presents. Children with CP included in this study varied widely in involvement, age, and level of spasticity (Table 1-S1). Yet, our exploratory analysis did not reveal differences in gains between hemiplegic and diplegic children (figure S6). Statistical testing for patient subgroups was not possible given the sample size. We visually explored whether gains were associated with age (figure S7-S10), body length (figure S11-S14), and gastrocnemius MAS (fig. S15-S16) but we did not observe any trends. Note that it has been hard to find associations between MAS and functional disabilities [34–36]. Similar factors could also not explain differences in the number of completed perturbation levels. Alternative factors might have contributed to variability in the response to balance perturbations. For example, biomechanical alignment during standing, which is often

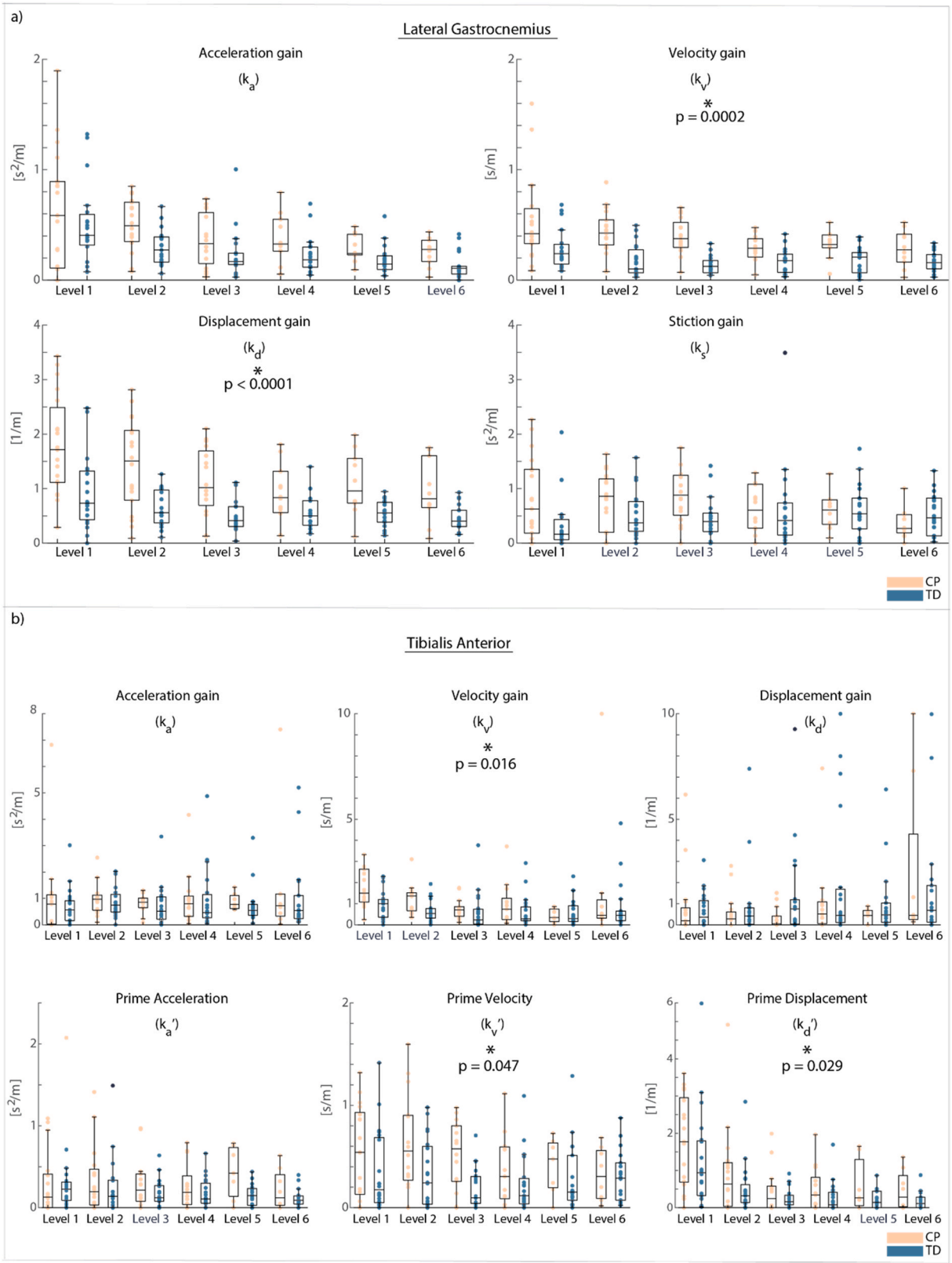

Fig. 5. CoM feedback gains ((prime) acceleration, (prime) velocity, (prime) displacement, and stiction) for all levels for the lateral gastrocnemius (top panel) and tibialis anterior (bottom panel). Differences between children with cerebral palsy (CP, orange) and typically developing (TD, blue) children were larger for lateral gastrocnemius (LG) than for soleus (SOL) and medial gastrocnemius (MG), therefore results for LG are shown in the main figure but figures for MG (Fig. S2) and SOL (Fig. S3) can be found in supplementary material (S5). Boxplots in black indicate median and interquartile ranges, dots are individual scores. Significant differences ($p < 0.05$) between groups are indicated with a star and p-values.

altered in CP, might contribute to altered muscle responses [37]. Also, differences in sensory deficits might have influenced balance performance. There is thus a need to investigate the origins of the variable balance impairments in CP.

We cannot exclude other hypotheses about sensorimotor transformations based on our results. The CoM feedback model was chosen based on previous research showing the ability of this model [14–18] as well as the inability of alternative models, e.g., feedback from head kinematics [38] or joint kinematics [14], to fit reactive muscle activity across conditions in healthy humans and animals. Nevertheless, CoM movement and plantarflexor muscle stretch in response to support-surface translations are correlated. Therefore, we cannot solely attribute the increased gains to altered balance correcting processes. Possibly, exaggerated stretch reflexes due to spasticity contributed as well. We plan to analyze the response to rotational perturbations to dissociate alterations in balance-correcting responses from alterations in the response to muscle stretch.

5. Conclusion

Sensorimotor pathways underlying balance control might be preserved in children with CP, but sensitivities are altered. The abnormal sensorimotor feedback might hinder balance control and explain stiffer balance responses in CP. The applied sensorimotor model provides insight into changes in neural processes in CP and might help diagnose balance impairments.

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CRediT authorship contribution statement

Jente Willaert: Recruitment; Data curation; Formal analysis; Methodology; Funding acquisition; Project administrator; Visualization; Writing – review & editing. **Giovanni Martino:** Validation; Writing – review & editing. **Kaat Desloovere:** Funding acquisition, Resources; Supervision; Writing – review & editing. **Anja Van Campenhout:** Recruitment; Resources; Supervision; Writing – review & editing. **Lena H. Ting:** Conceptualization; Funding acquisition; Methodology; Supervision; Writing – review & editing. **Friedl De Groote:** Conceptualization; Formal analysis; Funding acquisition; Investigation; Methodology; Resources; Supervision; Visualization;Writing – review & editing.

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Conflict of interest statement

None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.gaitpost.2023.03.014.

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